09/147693

WEST Search History

DATE: Tuesday, May 27, 2003

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L8	l3 same l4 same l5 same L7	83	L8
L7	mutat$	62110	L7
L6	l3 with l4 with l5	351	L6
L5	temperature	2958714	L5
L4	repress$	23191	L4
L3	l1 or L2	109026	L3
L2	phage	29556	L2
L1	lambda	96172	L1

END OF SEARCH HISTORY

WEST

Print Selection

Help | Clear | Cancel | Print | Print First Page

Select?	Document ID	Section(s)	Page(s)	# Pages to print	Database
☑	5789188	all	all	27	USPT,PGPB,JPAB,EPAB,DWPI
☑	4637980	all	all	9	USPT,PGPB,JPAB,EPAB,DWPI

Building	Room	Printer
cm1	11e14	gbldptr

Main Menu | Logout

=> s phage
L1 128436 PHAGE

=> s lambda
L2 248839 LAMBDA

=> s l1 or l2 s repress?
MISSING OPERATOR L2 S REPRESS?
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s temperature
L3 1761590 TEMPERATURE

=> s s repress
<------User Break------>

SEARCH ENDED BY USER
4 FILES SEARCHED...
SEARCH ENDED BY USER

=> s repress
L4 14802 REPRESS

=> s l1 or l2

L5 349570 L1 OR L2

=> s mutat?
L6 988137 MUTAT?

=> s l3 and l4 and l5 and l6
L7 2 L3 AND L4 AND L5 AND L6

=> s l3 and l5 and l6
L8 2251 L3 AND L5 AND L6

=> s l4 and l5 and l6
L9 76 L4 AND L5 AND L6

=> d l7 ibib abs 1-2

L7 ANSWER 1 OF 2 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:584887 BIOSIS
DOCUMENT NUMBER: PREV200200584887
TITLE: The regulation of hilA expression through the control of the negative regulator hilE.
AUTHOR(S): Baxter, M. (1); Jones, B. D. (1)
CORPORATE SOURCE: (1) University of Iowa, Iowa City, IA USA
SOURCE: Abstracts of the General Meeting of the American Society for Microbiology, (2002) Vol. 102, pp. 70. http://www.asmusa.org/mtgsrc/generalmeeting.htm. print. Meeting Info.: 102nd General Meeting of the American Society for Microbiology Salt Lake City, UT, USA May 19-23, 2002 American Society for Microbiology . ISSN: 1060-2011.
DOCUMENT TYPE: Conference
LANGUAGE: English
AB Salmonella invasion of host cells is dependent on proteins encoded by Salmonella Pathogenicity Island 1 (SPI-1). These genes, which encode many of the secreted effector proteins as well as the structural components of the type III secretion needle, are tightly regulated by the bacteria. Activation of these genes is dependent on environmental signals such as low oxygen concentration, high osmolarity, ***temperature*** and midlog growth. The central transcriptional activator of these genes is hilA. Our studies have identified a repressor of hilA known as hilE. This gene, located in a novel region of the Salmonella genome has been shown to ***repress*** hilA expression and Salmonella invasion when it is overexpressed. A ***mutation*** in hilE leads to increased expression of hilA and Salmonella invasion as measured by B-galactosidase activity and in vitro HEp-2 invasion assay, respectively. Additional work has shown that hilE is a Salmonella specific gene requiring a Salmonella specific factor for its expression. Current efforts are aimed at understanding how hilE exerts its effect on hilA expression and the signals that lead to the activation of hilE. Motifs found within hilE suggest that the protein is capable of binding to the hilA promoter thereby leading to the repression of hilA expression. This hypothesis is being investigated through the use of gel shift assays and challenge ***phage*** experiments. Activation of hilE appears to be a complicated process due to the presence of more than one activator that induces hilE expression. Future work will be aimed at identifying hilE regulators and characterizing how these regulators control the Salmonella invasive phenotype.

L7 ANSWER 2 OF 2 MEDLINE
ACCESSION NUMBER: 77252442 MEDLINE
DOCUMENT NUMBER: 77252442 PubMed ID: 330875
TITLE: Effects of ***mutations*** in the immunity system of bacteriophage P1.
AUTHOR: D'Ari R
SOURCE: JOURNAL OF VIROLOGY, (1977 Sep) 23 (3) 467-75. Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197710
ENTRY DATE: Entered STN: 19900314
Last Updated on STN: 19900314
Entered Medline: 19771020
AB A mutant of bacteriophage P1 that made an altered c1 repressor is described. The mutant c1 product had two configurations: in lysogens, at high temperatures, it permitted constitutive expression of the normally repressed DNA replication function ban and was insensitive to the action of ant, a product expressed by the virulent mutant P1 virs and by the heteroimmune ***phage*** P7 (formerly phiamp+) and normally able to overcome c1 repression; in mutant lysogens at low temperatures, the mutant repressor was apparently normal (able to ***repress*** ban and sensitive to ant action). Genetic studies of this mutant led to the isolation of a derivative that formed unstable lysogens. These studies suggested that the ban product was normally under c1 control; they further showed that ant overcame c1 repression by inactivating c1 rather than by creating a bypass of repressor activity.

=> d his

(FILE 'HOME' ENTERED AT 10:38:59 ON 27 MAY 2003)

FILE 'BIOSIS, EMBASE, MEDLINE, WPIDS, HCAPLUS' ENTERED AT 10:39:09 ON 27
MAY 2003
L1 128436 S PHAGE
L2 248839 S LAMBDA
L3 1761590 S TEMPERATURE
L4 14802 S REPRESS
L5 349570 S L1 OR L2
L6 988137 S MUTAT?
L7 2 S L3 AND L4 AND L5 AND L6
L8 2251 S L3 AND L5 AND L6
L9 76 S L4 AND L5 AND L6

=> dup rem l9
PROCESSING COMPLETED FOR L9
L10 40 DUP REM L9 (36 DUPLICATES REMOVED)

=> s l10 and py<1997
1 FILES SEARCHED...
3 FILES SEARCHED...
4 FILES SEARCHED...
L11 27 L10 AND PY<1997

=> s repress?
L12 164198 REPRESS?

=> s temperature?
L13 1896541 TEMPERATURE?

=> s l5 and l12 and l3 and l6
L14 255 L5 AND L12 AND L3 AND L6

=> dup rem l14
PROCESSING COMPLETED FOR L14
L15 155 DUP REM L14 (100 DUPLICATES REMOVED)

=> s l15 and py<1997

1 FILES SEARCHED...
3 FILES SEARCHED...
4 FILES SEARCHED...
L16 131 L15 AND PY<1997

=> d l16 ibib abs 1-131

L16 ANSWER 1 OF 131 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1997:13123 BIOSIS
DOCUMENT NUMBER: PREV199799312326
TITLE: A procedure for the prediction of ***temperature*** -sensitive mutants of a globular protein based solely on the amino acid sequence.
AUTHOR(S): Varadarajan, R. (1); Nagarajaram, H. A.; Ramakrishnan, C.
CORPORATE SOURCE: (1) Molecular Biophysics Unit, Indian Inst. Sci., Bangalore 560 012 India
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1996) Vol. 93, No. 24, pp. 13908-13913.
ISSN: 0027-8424.
DOCUMENT TYPE: Article
LANGUAGE: English
AB ***Temperature*** -sensitive (Ts) mutants of a protein are an extremely powerful tool for studying protein function in vivo and in cell culture. We have devised a method to predict those residues in a protein sequence that, when appropriately ***mutated*** , are most likely to give rise to a Ts phenotype. Since substitutions of buried hydrophobic residues often result in significant destabilization of the protein, our method predicts those residues in the sequence that are likely to be buried in the protein structure. We also indicate a set of amino acid substitutions, which should be made to generate a Ts mutant of the protein. This method requires only the protein sequence. No structural information or homologous sequence information is required. This method was applied to a test data set of 30 nonhomologous protein structures from the Protein Data Bank. All of the residues predicted by the method to be gtoreq 95% buried were, in fact, buried in the protein crystal structure. In contrast, only 50% of all hydrophobic residues in this data set were gtoreq 95% buried. This method successfully predicts several known Ts and partially active mutants of T4 lysozyme, ***lambda*** ***repressor*** , gene V protein, and staphylococcal nuclease. This method also correctly predicts residues that form part of the hydrophobic cores of ***lambda*** ***repressor*** , myoglobin, and cytochrome b562.

L16 ANSWER 2 OF 131 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1996:186808 BIOSIS
DOCUMENT NUMBER: PREV199698742937
TITLE: C-terminal deletions can suppress ***temperature*** -sensitive ***mutations*** and change dominance in the ***phage*** Mu ***repressor*** .
AUTHOR(S): Vogel, Jodi L.; Geuskens, Vincent; Desmet, Lucie; Higgins, N. Patrick (1); Toussaint, Ariane
CORPORATE SOURCE: (1) Dep. Biochemistry Molecular Genetics, Univ. Alabama, 861-A Bevill Biomedical Res. Build., Box 13, 845 19th St. South, Birmingham, AL 35294-2170 USA
SOURCE: Genetics, (1996) Vol. 142, No. 3, pp. 661-672.
ISSN: 0016-6731.
DOCUMENT TYPE: Article
LANGUAGE: English
AB ***Mutations*** in an N-terminal 70-amino acid domain of bacteriophage Mu's ***repressor*** cause ***temperature*** -sensitive DNA-binding activity. Surprisingly, amber ***mutations*** can conditionally correct the heat-sensitive defect in three mutant forms of the ***repressor*** gene, cts25 (D43-G), cts62 (R47-Q) and cts71 (M28-1), and in the appropriate bacterial host produce a heat-stable Sts phenotype (for survival of ***temperature*** shifts). Sts ***repressor*** mutants are heat sensitive when in supE or supF hosts and heat resistant when in Sup degree host. Mutants with an Sts phenotype have amber ***mutations*** at one of three codons, Q179, Q187, or Q190. The Sts phenotype relates to the ***repressor*** size: in Sup degree hosts sts ***repressors*** are shorter by seven, 10, or 18 amino acids compared to ***repressors*** in supE or supF hosts. The truncated form of the sts62-1 ***repressor*** , which lacks 18 residues (Q179-V196), binds Mu operator DNA more stably at 42 degree in vitro compared to its full-length counter-part (cts62 ***repressor***). In addition to influencing ***temperature*** sensitivity, the C-terminus appears to control the susceptibility to in vivo Clp proteolysis by influencing the multimeric structure of ***repressor*** .

L16 ANSWER 3 OF 131 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1996:66952 BIOSIS
DOCUMENT NUMBER: PREV199698639087
TITLE: Regulation of the heat-shock response depends on divalent metal ions in an hflB mutant of Escherichia coli.
AUTHOR(S): Herman, Christophe; Lecat, Sandra; D'Ari, Richard; Bouloc, Philippe (1)
CORPORATE SOURCE: (1) Inst. Genet. Microbiol., Univ. Paris-Sud, CNRS/URA 1354, Batiment 400, 91 405 Orsay Cedex France
SOURCE: Molecular Microbiology, (1995) Vol. 18, No. 2, pp. 247-255.
ISSN: 0950-382X.
DOCUMENT TYPE: Article
LANGUAGE: English
AB HflB, also called FtsH, is an essential Escherichia coli protein involved in the proteolysis of the heat-shock regulator sigma-32 and of the ***phage*** regulator ***lambda*** -cII. The hflB1(Ts) allele (formerly called ftsH1) conferring ***temperature*** -sensitive growth at 42 degree C is suppressed by loss of the ferric-uptake ***repressor*** Fur and by anaerobic growth. We show here that suppression requires TonB-dependent Fe(III) transport in the hflB1(Ts) fur mutant during aerobic growth at 42 degree C and Feo-dependent Fe(II) transport during anaerobic growth at 42 degree C. ***Temperature*** -resistant growth of hflB1(Ts) strains is also observed at 42 degree C in the presence of a high concentration of Fe(II), Ni(II), Mn(II) or Co(II) salts, but not in the presence of Zn(II), Cd(II), Cu(II), Mg(II), Ca(II) or Cr(III) salts. However, neither Ni(II) nor a fur ***mutation*** permits growth in the complete absence of HflB. The heat-shock response, evaluated by an htpG::lacZ fusion, is overinduced in hflB1(Ts) strains at 42 degree C because of stabilization of sigma-32. Growth in the presence of Ni(II) or in the absence of the Fur ***repressor*** abolishes this overinduction in the hflB1(Ts) strain, and, in the hflB1(Ts) fur mutant, sigma-32 is no longer stabilized at 42 degree C. These results reinforce the recent observation that HflB is a metalloprotease active against sigma-32 in vitro and suggest that it can associate functionally in vivo with Fe(II), Ni(II), Mn(II) and Co(II) ions.

L16 ANSWER 4 OF 131 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1995:297846 BIOSIS
DOCUMENT NUMBER: PREV199598312146
TITLE: Control of lytic development in the Streptomyces temperate ***phage*** vphi-C31.
AUTHOR(S): Wilson, Stuart E.; Ingham, Colin J.; Hunter, Iain S.; Smith, Margaret C. M. (1)
CORPORATE SOURCE: (1) Dep. Genetics, Queens Med. Centre, University Park, Nottingham NG7 2UH UK
SOURCE: Molecular Microbiology, (1995) Vol. 16, No. 1, pp. 131-143.
ISSN: 0950-382X.
DOCUMENT TYPE: Article
LANGUAGE: English
AB The ***repressor*** gene, c, is required for maintenance of lysogeny in the Streptomyces ***phage*** vphi-C31. The c gene expresses three in-frame N-terminally different protein isoforms at least one of which is